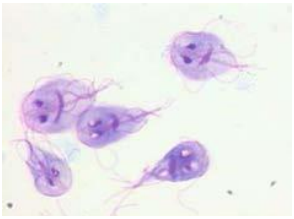
Gastrointestinal Microbiology

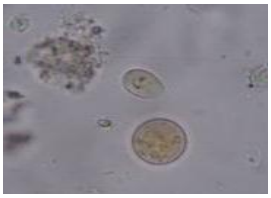
Abdelrahman Ashour

Giardia lambila

Characteristics:-it has 2 form



1-Trophozoite:-2 nuclei and central parabasal bodies give it the appearance of a face with two eyes(زي شكل البومة)with 4 pair of Flagella(8 flagella) and we can find it mainly in duodenum and Jejunum which cause pathological changes there



2-cyst(infective form):-it has 4 nuclei or more,it develop inside colon and it has high Resistance from cold,high T and high Chloride concentration in the water

بتتحول مباشرة من trophozoiteالىcystاول ما تدخل القولون لانها عارفة انها رايحة على بيئة صعبة بالخارج

Route of transmision:-fecal-oral route(by poorly purified water mainly, food, in day care)

Disease/Pathogenesis:-this parasite will cause "Giardisis" and it cause it by attached superficially(without invasion) to brush border of small intestine which will cause destruction of microvili and malabsorbtion

Clinical symptoms:foul-smelling, fatty stool-steatorrhea-, malabsorbtion of DEKA vitamins(A,K,E,D),vit-B12,fat&CHO (lactose intolerance with gastric distention),watery diarrhea(not bloody) and low-grade fever

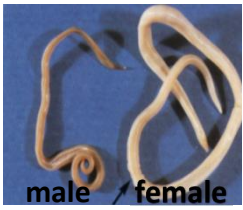
Risk factor:-1)IgA deficiency /2)contaminated water in developing counteries /3)achlorhydria or hypochlorhydria in patient

Diagnosis:-1)stool analysis to see cyst or trophozoite by microscope
2) EIA to detect G.lambila Ag in the stool

Treatment:-first choice is metronidazole but in pregenant women we use Paromomycin------->no vaccine!

Round worms(Nematodes)

General Characteristics:-Non-segmented,cylindrical worms tapering at both ends,small size(1cm-100cm),Sexes are separate, male is smaller than female & its posterior end of male is curved ventrally



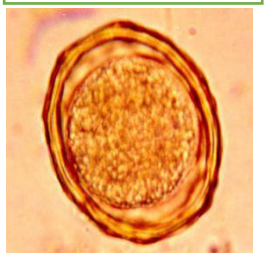
Modes of transmession:-
1-Ingestion of embryonated eggs from contaminated food & drinks (ex:A.lumbricoides&Trichinella spiralis)
2-Penetration of skin(ex:S.stercoralis)
3-blood sucking insects(ex: filarial worms)
4-Inhalation of infected dust(ex:E.vermicularis)

Round worm classification:-
1-round worm which live in small intestine only(ex:Ascaris lumbricoides,hook worm &american hook worm…..etc)
2-Round worm which live in appendix and caecum(ex:Enterobius vermicularis (pin worm),Trichuris trichiura (whip worm))

We have 5 species of nematodes:-

1- Ascaris lumbricoides(roundworm):-most common helminth worldwide; largest roundworm ("Lumbering Tree Man in sketch")

Embryonated eggs



Characteristics of it:-male smaller than female,egges isbile stained Albuminous(thick) coat with unsegmented ovum

Mode of transmission:Ingestion of eggs(fecal-oral route) and it is localized in small intestine

infective form:Embryonated eggs

Life cycle:-Eggs are ingested in small intestine then pentrate the wall of it to travel through blood-->enter lungs-->then go back to GI tract

Disease/ Organs Most affected: it is caused disease which called Ascariasis which affect mainly small intestine

Symptoms & Complications:-
A-by migrating larvae:
1-Pneumonia (loeffler's syndrome):bloody sputum that may contain larva, urticarial rash & eosinophilia
2-Visceral larva migrans:if larvae enter systemic circulation (from pulmonary capillaries) to reach other organs like brain, spinal cord, heart, kidney

B-by adult worm:
1-diarrhoea,Protein energy malnutrition (PEM),Vit. A deficiency (night blidness),Intestinal obstruction,perforation – peritonitis, hypersynsetivity reaction.
2-Ectopic Ascariasis(migration of worm up into the stomach or any where in the body like larynx,nose,mouth,appendix(appendicitis)…

Diagnosis:-
A-Microscopic direct examination of feces following floatation method: bile stained eggs.
B-Blood examination --->eosinophilia
C-Macroscopic examination(see on stool or vomit بنشوفهم بعيننا المجردة)

Treatment:-first choice is Mebendazole/ Albendazole(not used in pregnancy or heavy infection),and we also can use Pyrantel pamoate

Prevention:-mass treatment by give mebendazole as prophlaxisis

2-Ancylostoma duodenale(hook worm) and Necator americanus (american hook worm):-"american hero with hook in sketch"

Characteristics of it:-male smaller than female,eggs are non bile stained (colorless),segmented with 4blastomeres

Infective form:3rd stage filariform larva

Mode of transmission:Penetration into skin(especially the Thin skin between toes,Dorsum of the feet&Inner side of the soles)and then go to small intestine

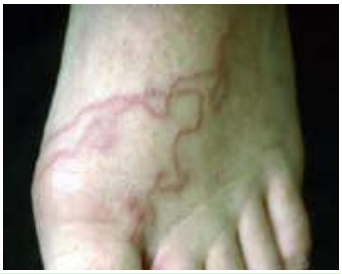
Risk factor:-people who is at risk are Gardeners & miners

Life cycle:filariform larvae--->penetrate the skin--->circulation---> lung--->trachea--->pharynx----->swallow it and reach intestine to mature there(adult worm)----->eggs formation inside the small intestine--->eggs in feces

Disease and symptoms:-

A- by larvae:

1-Ancylostome dermatitis or Ground itch(more common in necator occur at site of penteration)
2-Creeping eruption – reddish itchy papule
3-in the lungs – bronchitis & bronchopneumonia



Creeping eruption

B- by adult worm:

1-Epigastric pain, diarrhoea & vomiting
2-Microcytic hypochromic(Iron deficiency) anaemia – due to chronic blood loss(worm sucks blood and bleeding from pentrating site)

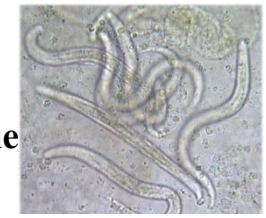
Diagnosis:

1-microscopic examination of stool(non-bile stained,segamented egg)
2-Occult blood test is positive
3-Blood examination----->anaemia+eosinophilia

Treatment:-first choice is Mebendazole/ Albendazole(not used in pregnancy or heavy infection),and we also can use Pyrantel pamoate and we should be give oral iron supplement to treat iron deficiency anaemia

Prevention:good hygiene and mass treatment by give mebendazole as prophlaxisis

3-Strongyloides stercoralis:it is the smallest one"strong guy in sketch"



Characteristics of it:adult worm(2-2.5mm) mostly found living in moist soil &when get inside the humen body put it eggs within tissue

Infective form:Filariform larvae

Mode of transmission:Penetration of skin / autoinfection
***autoinfection means same person infect itself if it has very poor hygiene يعني بعطيه دواء وبتعالج بس لما يدخل الحمام ويغسل ما بغسل منيح برجع بعدي حاله**

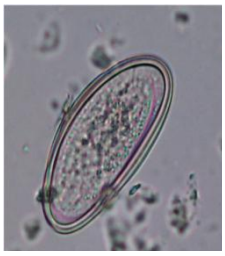
Life cycle:filariform larvae--->penetrate the skin--->circulation---> lung--->trachea--->pharynx---->swallow it and reach to wall of small intestine(mainly duodenum & jejunum)---->then put eggs within intestinal tissue---->eggs will hatch and give rhabditiform larva----> rhabditiform larva will leave the body within faeces--->rhabditiform larva will mature to filariform larvae within moist soil outside the body

Disease and symptoms:-
1-skin lesion"larva currens":-urticarial rash&erythematous in site of entery and perianal region
2-Pulmonary lesions(due to migrating larva):will cause Alveolar hemorrhages&Bronchopneumonia
3-Intestinal lesions"burrowing lesions" :Epigastric pain,Diarrhoea with blood & mucus,Nausea & Weight loss

Diagnosis:
1-Stool examination by see rhabditiform larva in stool
2-Culture(not common use)
3-Serological test(not common use)

Treatment:Thiabendazole for 2 days&Ivermectin

4-Enterobius vermicularis(pin worm/seat worm)



Characteristics of it:male smaller than female,eggs is 60 µm non bile stained Plano-convex(D-shaped) with coiled embryo

Infective form:Embryonated egg

Mode of transmission:fecal-oral (Ingestion),Autoinfection

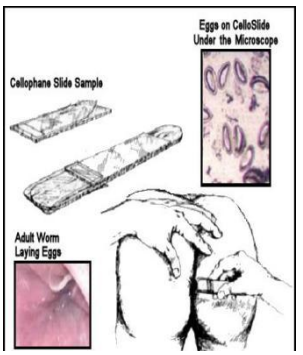
Life cycle: Embryonated egg ingest by mouth then go to small intestine to hatch to larvea----->larvea will mature to adult worm inside Large intestine(caecum & appendix) to cause infection

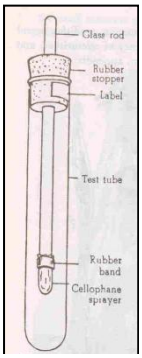
Disease & symptoms:
1-Perianal, perineal & vaginal itching (pruritis) worsens at night(Due to migration of worm to GU)
2-Insomnia(due to nocturnal GU itching)
3-Nocturnal enuresis(تبول لا ارادي ليلا)

Diagnosis:
1-Detection of adult worms in Feces &Perianal region
2-NIH swab scrapings from perianal region
3-Microscopy – non bile stained eggs

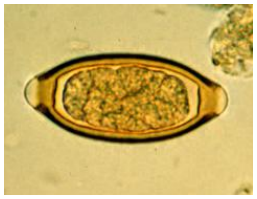
Treatment: Mebendazole, pyrantel pamoate

NIH swab

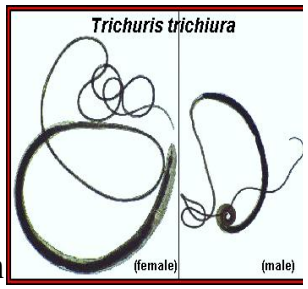




5-Trichuris trichiura(Whip Worm):("Porky Trickster wearing a spiral suit in sketch")



Characteristics of it:male smaller than female,eggs are bile stained Barrel-shaped with Mucus plug at each pole with Unsegmented ovum"tray shape"شكلها زي الصينية
And we called it whip worm because it has whip"سوط" at end



Infective form:mature embryonated egg

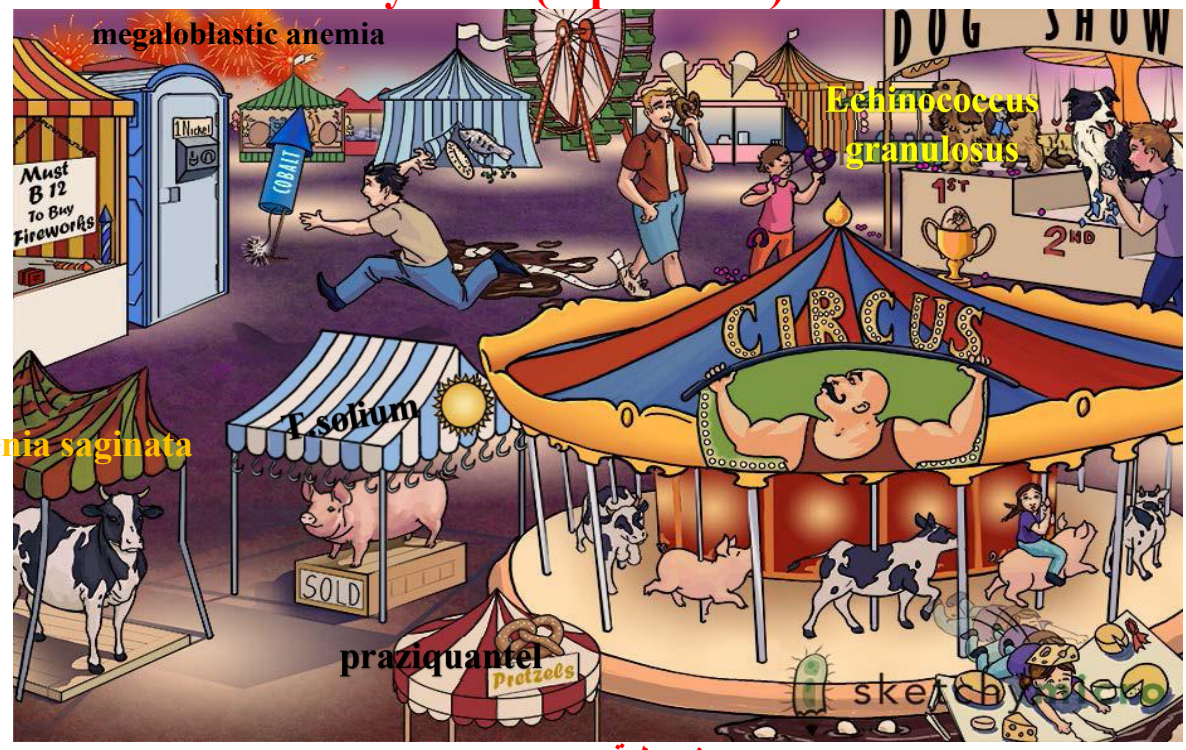
Mode of transmission:fecal-oral (Ingestion)

Life cycle: Embryonated egg ingest by mouth then go to small intestine to hatch to larvea----->larvea will mature to adult worm inside Large intestine(caecum) to cause infection

Disease and symptoms:
1-we called it's disease Trichuriasis(mainly asymptomatic if worm Less than 10 in number)
2-profuse mucus and bloody diarrhea with abdominal pain, weight loss and anemia

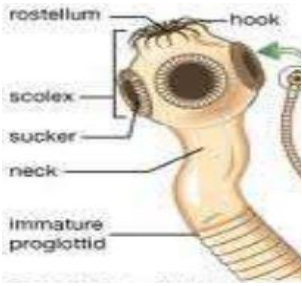
Diagnosis &treatment: Stool examination to see embryonated eggs and treat this infection by albendazole / mebendazole

Cystodes(tape worm)



General Characteristics:-long, ribbonشريطية**-like helminths, segemented tape with hermaphroditic (**خنثى**male &female in same body segement)unit releasing eggs via rupture or through uterine pore,and it's body is lack from vascular,respiratory systems,gut and body cavity&cause fast weight loss**

It's body contain 3 main parts:scolex(hook &sucker structures to help it to absorb nutrients from the host's GI tract) neck, and segmented body parts



We have 3 important species of cestode:-

1-Taenia saginata:-"tent above cow in sketch"

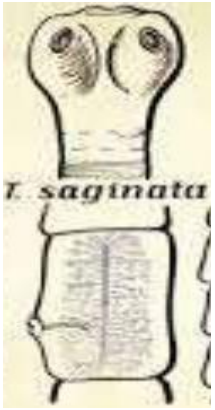
Characteristics of it:-

1-The eggs are 30-40mm in diameter bile stained(dark color) , solid rounded shell(thick radiated wall) and contain a fully developed, six-hooked (hexacanth) embryo

2-has Scolex with 4 suckers

3-the segment is beging from head as large,wider&more mature Segment then become smaller and detachement easily at end of tail

4-it might be reach 10m in length with about 100,000 eggs in each segment



Infective form:-cysticercus

Mode of transmission:ingestion of cyst

Intermediate host(HI):-cattle(cow)/definitive host(DH):-humen

Life cycle:-eggs from infective person will drop in the stool---->then the cattle(intermediate host) will drink the contaminated water with eggs------>eggs will enter the cattle body and then mature to cyst within it's striated muscles tissue------>if the another person eat inadequately cooked cattel meat which contain the cyst,it will enter the humen body and mature to adult larvea worm which localized in jejunum to cause intestinal infection

Disease&symptoms:-
1-BEEFTAPE WORM DISEASE:-which called it also Taeniasis saginata which is asymptomatic or mild disease because the humen here isn't intermediate host and we just know it by spontaneous passage of proglottids in stool
2-epigastric discomfort, nausea, irritability, diarrhea, and weight loss

Diagnosis:-
1-stool examination to see eggs or proglottides(segment)
2-cellophane tape technique:by put cellophane tape at anal ring at night and this method is more effective than stool examination

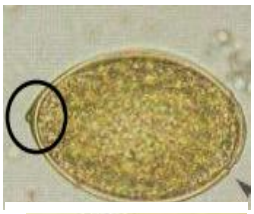
Treatment:-drug of choice is praziquantel and also we can use niclosamide

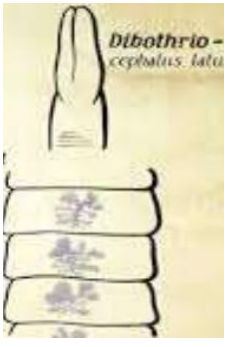
Prevention:-
1-cooked the meat in 56 C° for 5 min
2-Salting or freezing meat

Note:Taenia solium has the same Characteristics of taenia saginata but the T.solium has hook on there head and the intermediate host of it is swine(pig) not cattle

---

2-Diphyllobothrium latum"bathroom in the scetch":it is the longest tapeworm





Characteristics of it:-
1-eggs are Oval shape,Thin wall,Colorless,
Operculum (بروز صغير على طرف البيضة)with nondeveloped-immature-embryo inside of it
2-Scolex like crocodile تمساح
3-Wider-broad- & shorter proglottids(segments)

Infective form:-cyst(big enough to see by eye)

Mode of transmission:-ingestion of contaminated fish with cyst

Intermediate host(IH):-it is require 2 intermediate host to complete it's life cycle ,copepodسلطون &freshwater fish

Defentive host(DH):-humen

Life cycle:-immature-eggs from infective person will drop in the stool--->it will reach the water and convert to mature eggs inside it---->then coracidia will hatch from mature eggs in water and ingest by copepod(Cyclops or Diaptomus)---->the coracidia will convert to procercoid inside the copepod tissue---->fish will eat infective copepod and the prococercoid will convert to plerocercoid within musculature fish--->then humen will eat improperly prepared freshwater fish---->D.latum will reach the small intestine and will attach to ileal mucosa with 2 sucking groove

Disease&symptoms:-
1-FISH TAPE WORM DISEASE:Most infected patients are asymptomatic
2-epigastric pain, abdominal cramping, vomiting, and weight loss
3-intestinal or biliary obstruction(due to several adult large size)
4-Macrocytic(megaloblastic) anemia and vitamin B12 deficiency is related to the consumption by the worm

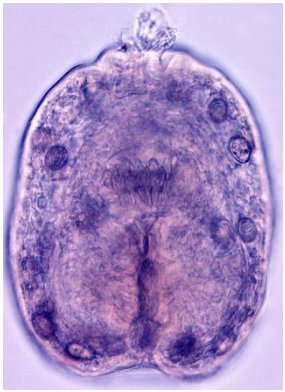
Diagnosis:-simply be see proglottids(segments) by eye without microscope(because it big enough)

Treatment:-
1-drug of choice is praziquantel and also we can use niclosamide
2-When anemia or neurologic manifestations are present, parenteral administration of vitamin B12

Prevention:-proper cooked of fish or put the fish in 10 C° for 48h

---

3-Echinococcus granulosus"dog on 1st stage in sketch":-it is the shortest tapeworm



Characteristics of it:-
1-embryonated eggs are radial striated wall
2-adult worm has hook with elongated segment
3-It's length reach just 0.005 m(very short)

Infective form:-hydatid cyst

Mode of transmission:-fecal-oral route(from dog feces)

Intermediate host(IH):-humen or sheep

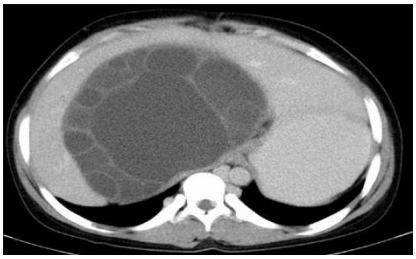
Defentive host(DH):-dogs/wolves

Disease &sympotms:-
1-if the humen become DH the cyst will hatch to adult worm inside the GI to cause localize infection in the GIT(diarrhea,wt loss..)

2-But if the humen become IH(in immunocompromised) the symptoms will be very severe because the adult worm will reach the liver by portal vien and convert to cyst which may cause the liver failure(due to large amount of cyst in the liver)

3-Many cyst(30-40) in liver,lung and kidney

Diagnosis:-
1-blood examination---->eosinophilia
2-imaging by CT to see cyst in the liver



Treatment:-Surgery to remove cyst from liver &albendazole

For Hymenolepis nana just to know the DH&IH is humen and it polar filaments embryonated eggs with short broad segment body

==========

METAZOAN FLAT WORM(TREMATODE FLUKES)

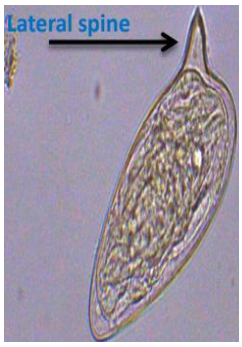
SCHISTOSOMA

General Characteristics:-leaf shaped worms which are generally flat and fleshy, are hermaphroditic except for *Schistosoma*, which are separate male and female organ in one body; have complicated life cycles occurring in two or more hosts,have operculated eggs (except for *Shistosoma*),shistosoma inhabit the portal vascular system of a number of animals

We have 3 groups of shistosoma depend on site of infection:-

1-Schistosoma mansoni &Schistosoma intercalatum ما نسوني لاني كلمتهم

Characteristics of it:-The eggs of S. mansoni are oval, possess a sharp lateral spine, and measure 60-140 µm



Endemic erea:-Eygpt,Iraq&sudan(South America,Africa, and the Middle east) but S.intercalatum found mainly in central west africa

Intermediate host:-snails defentive host:-humen

Mode of transmission:-penetration of skin

Infective form:-cercariae

Disease:-cause intestinal schistosomiasis

Life cycle:-mating of the adult worms in the portal vein, then ascend to the mesenteric vessels against the flow of blood---->S. mansoni are directed to the inferior mesenteric system---->then coming to rest in the venous plexus of the bladder and other pelvic organs--->then reaching the submucosal venules,to start oviposition(put 300 eggs)--->ova(egg) will drop in the bowel then in the stool---->ova will reach the fresh water and hatch to miracidia--->the miracidia will penetrate the snails to develop into it to forked-tailed cercariae in 2month---->cercariae will penetrate the humen skin--->from the skin to small vanules then to right side of heart then to lung in 3 days----> from the lung throughout the trachea reach gut---->from gut to portal vein to mating in it for 3 month

Life span:4-35 years

Epidemiology of the disease:-helminthic infection in the world today which kill 1 million annually(high morbidity)

Immunity and clinical manifestation:-
1-Early stage:-delayed hypersensetivity type 4 by form granuloma around penetration site on skin to cause pruritic papular skin rash with severe itching and then it will travel to liver and cause GIT symptoms(fever,abdominal pain,headach) with elevation in serum IgG4

2-Intermidiate stage:-after 2 month the disease will become more severe and cause type 3 hypersentivity-serum sickness- which cause many pathological change such as fever and chills, patients experience cough, urticaria, arthralgia, lymphadenopathy, splenomegaly, abdominal pain, and bloody diarrhea &some of Ag-Ab complex(type 3 hypersensetivity)might reach systemic circulation to cause glomerulonephritis(in kidney) and epilepsy or paraplegia(in CNS)

3-chronic stage:- retained eggs which not drop in the stool will induce Inflammatory and fibrotic reactions by Soluble antigens excreted by the eggs stimulate the formation of T lymphocyte–mediated eosinophilic granulomas to cause chronic disease
Note about chronic stage:-↑number of retained eggs--->↑severity of chronic diasease(high morbidity)

4-some of retained eggs it might back to the liver by portal vien to cause hepatomegally --> cirrhosis --->portal hypertension---> Ascites--->varices mainly in esophagus--->sometimes rupture of varices which will cause sever hematemesis then death

5-Schistosoma infection in general it might cause cancer(bladder or bowel cancer)

Diagnosis:-
1-CBC---->eosinophilia
2-stool analysis to see eggs
3-Liver function test: serum globulin rise, ALT slightly rise
4-serological test:-Ab for worm
5-cytoscopy&rectal biopsy

Treatment:-drug of choice is Praziquantel &may use Antihistamines and corticosteroids to treat immune reactions

Prevention:-by use Chemical molluscicides for water/NO vaccine!!!

2-S.haematobium(Bilharzia) مات منها عبد الحليم حافظ الله يرحمه

Characteristics of it:-The eggs of S.haematonium are oval, possess a sharp terminal spine, and measure 60-140 µm

Endemic erea:-Africa and the Middle East

Intermediate host:-snails **defentive host:-humen**

Mode of transmission:-penetration of skin

Infective form:-cercariae

Disease:-cause urinary schistosomiasis(bilharziosis)

Life cycle:-exactly like S.mansoni but with tiny different,in S.mansoni the egg will drop in bowel then leave the body by stool ,BUT in S.haematobium the egg will drop in the urinary bladder then leave the body by urine

Life span:4-35 years

clinical manifestation:-
1-bladder mucosa becomes thickened, papillated, and ulcerated
2-Hematuria and dysuria with anemia
3-loss of bladder capacity and contractibility
4-Uremia and renal failure
5-Bladder carcinoma is frequently seen

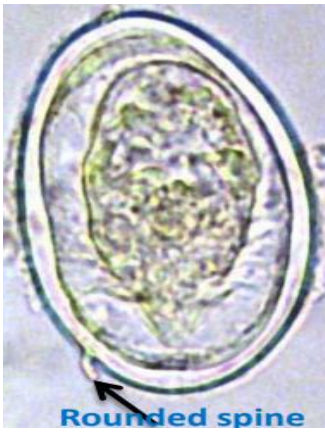
Diagnosis:-
1-CBC---->eosinophilia
2-urine analysis to see eggs
3-kidney test(urea and creatinine)
4-cytoscopy&bladder biopsy

Treatment:-drug of choice is Praziquantel &may use Antihistamines and corticosteroids to treat immune reactions

Prevention:-by use Chemical molluscicides for water/NO vaccine!!!

3-S.japonicum جابوني على اليابان

Characteristics of it:-The eggs are more nearly circular with a minute lateral rounded spine.measuring 70-90 µm.



Endemic erea:-Far East&Southeast Asia(japan)

Intermediate host:-snails defentive host:-humen

Mode of transmission:-penetration of skin

Infective form:-cercariae

Disease:-cause Asian intestinal schistosomiasis

Life cycle:-mating of the adult worms in the portal vein, then ascend to the mesenteric vessels against the flow of blood---->S. japonicum enters the superior mesenteric vein---->then reaching the venous radicals of the small intestine--->then to ascending colon; and end in the descending colon and rectum(then put 3000 egg in rectum) --->ova(egg) will drop in the bowel then in the stool---->ova will reach the fresh water and hatch to miracidia--->the miracidia will penetrate the snails to develop into it to forked-tailed cercariae in 2month---->cercariae will penetrate the humen skin--->from the skin to small vanules then to right side of heart then to lung in 3 days----> from the lung throughout the trachea reach gut---->from gut to portal vein to mating in it for 3 month

Life span:4-35 years

clinical manifestation:-exactly as S.mansoni but with more liver complication(jundice,portal hypertension…etc)

Diagnosis:-
1-CBC---->eosinophilia
2-stool analysis to see eggs
4-serological test:-Ab for worm
5-cytoscopy&rectal biopsy

Treatment:-drug of choice is Praziquantel &may use Antihistamines and corticosteroids to treat immune reactions

Prevention:-by use Chemical molluscicides for water/NO vaccine!!!

Viral diarrhea

General epidemiology of the disease:-

1-The most common cause of diarrhea is viruses(75% cases of acute diarrhea)

2-In developing countries,viral gastroenteritis(2nd most common viral illness after URTI) is a major killer of infant(2-5 Y) especially Rotavirus due to dehydration

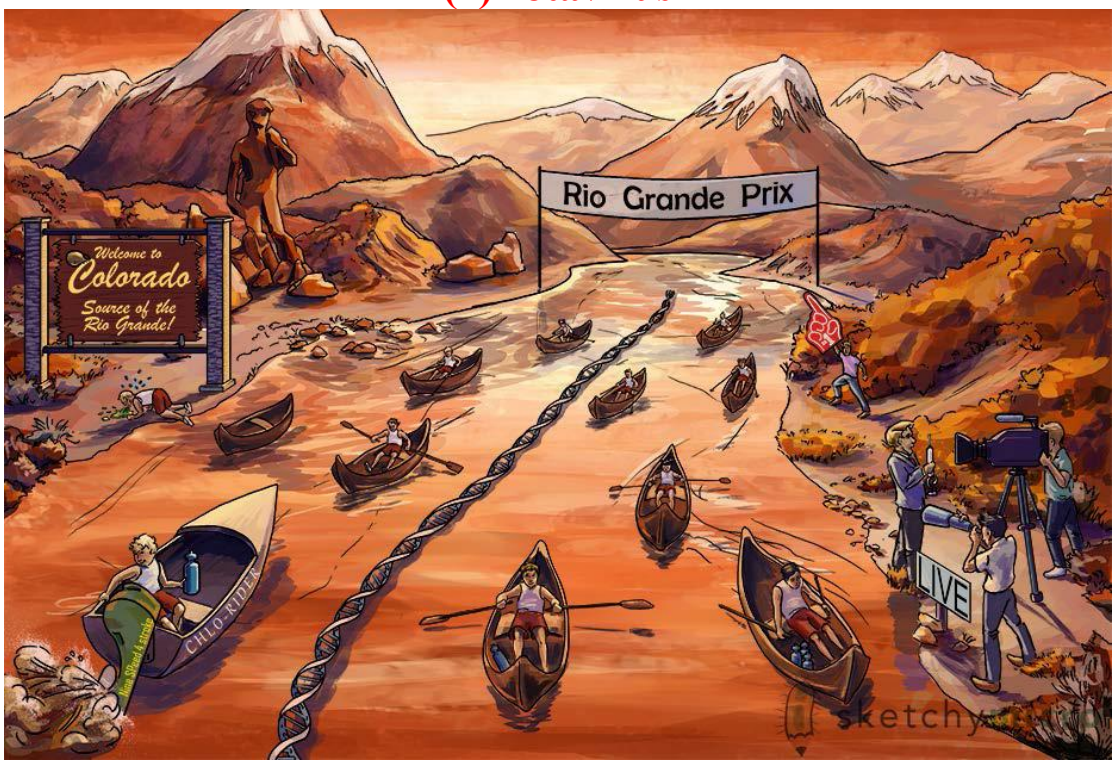
3-We need a huge number of virus(10^8) to see under the electron microscope but now we start to use PCR and EIA to detect viral infection

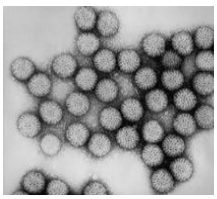
4-Virus mainly cause watery diarrhea

==========

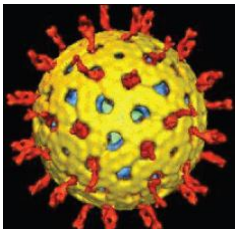
Causes of Viral Diarrhea:-we have many types of virus which cause diarrhea such as:-

(1)Rotavirus





Characteristics of it:-small in size(70 nm round),naked,double stranded RNA,11 segments of genome&Double capsid/shelled (outer and inner capsid) with wheal-radiating spokes to helps in attachment And also is replicate in cytoplasm



Risk factor:-increase risk of infection in winter and effect mainly the Children under 2 years

Pathogenic group for humen:-Group A subtypes 1, 2, 3, 4

Mode of transmission:-Fecal-oral route

epidemiology of the disease:-
1-#1 cause of severe infantile gastroenteritis(40-60%) that leads to watery diarrhea which occurring during the cooler months in infants and children less than 2 years of age but in adult the attack rates are usually much lower
2-most of people at 4 years old has 90% humeral antibody against rotavirus (natural immunazation)

Incubation period:-1-3 days

Disease&symptoms:-
1-Vomiting for 1-3 days
2-Diarrhea will Start with brown watery stools and then will convert to hakuri"white stool diarrhea" for 4-8 days
3-low grade fever
4-Severe complications is severe dehydration which lead to hypernatremia&hypo/hyper-kalemia

Diagnosis:-
1-stool analysis for viral Ag
2-PCR&EIA
3-Serum electrolytes

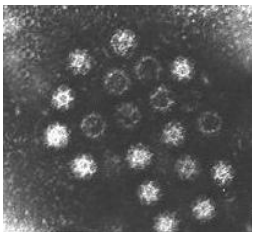
Treatment:-don't use drugs because it is self-limiting so give only supportive care(Symptomatic treatment) mainly oral rehydration to correct electrolytes imbalance

Prevention:-careful hand washing,adequate disposal of enteric excretions and take a vaccine at begininig of winter(has a vaccine)

==========

(2) Calicivirus

Characteristics of it:-Small +ssRNA viruses(35 nm in diameter smaller than rota),naked,icosahedral shape,consisting of hollows, positive-sense ssRNA



Pathogenic group for humen:-4 different serotype affect humen

People at risk:-older children and adults at any season(unlike rota)

Mode of transmission:-Fecal-oral route by consumption of contaminated water, uncooked shellfish, and other foods

epidemiology of the disease:-
1-the most common type of CALICIVIRIDAE family is Norovirus (Norwalk agent)
2-May cause outbreak at school or in same family الكل بنعدي فجأة
3-most of people at 50 years old has 50% humeral antibody against calicivirus (natural immunazation)

Incubation period:-10-51 hours(less than rota)

Disease&symptoms:-
1-clinical symptoms exactly like rota but the Vomiting here is prominent.
2-Respiratory symptoms rarely coexist.

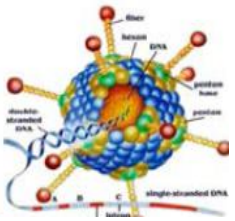
Diagnosis:-
1-microscopic examination to see virus under electron microscopy or immunoelectron microscopy
2-PCR&EIA
3-Serum electrolytes

Treatment:-don't use drugs because it is self-limiting so give only supportive care(Symptomatic treatment) mainly oral rehydration to correct electrolytes imbalance

Prevention:-good hygienic measures/no Vaccine!!

(3) Adenoviruses

Characteristics of it:-Small dsDNA viruses(75 nm in diameter little larger than rota),naked,icosahedral shape(hexons with pentons and fibers)



People at risk:-young children and neonates

Mode of transmission:-Fecal-oral route&respiratory droplets

epidemiology of the disease:-
1-#2nd most common viral cause of gastroenteritis (7-15%) after rota
2-Can cause occasional outbreaks
3-most of people at 3 years old has humeral antibody against adenovirus (natural immunazation)

Disease&symptoms:-exactly like rota

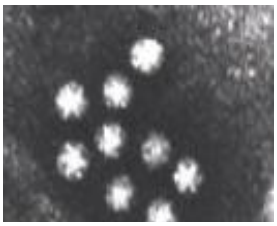
Diagnosis:-
1-microscopic examination to see virus under electron microscopy or immunoelectron microscopy
2-ELISA to detect viral Ag in stool

Treatment:-don't use drugs because it is self-limiting so give only supportive care(Symptomatic treatment) mainly oral rehydration to correct electrolytes imbalance

Prevention:-has live non-attenuated vaccine

(4) Astroviruses

Characteristics of it:-Small +ssRNA viruses(28 nm in diameter which is smallest virus),named because of star-shaped surface morphology, naked.سميناه الفيروس النجمي لانه فعلا تحت المجهر ببين فيه نجوم



People at risk:-infants&immunocompromized patient

Mode of transmission:-Fecal-oral route

epidemiology of the disease:-
1-Responsible for up to 10% of cases of gastroenteritis
2-Cause outbreaks, primarily among toddlers, school children and elderly nursing home residents
3-Most people have antibodies by the age of three

Disease&symptoms:-Similar disease to rota and adenoviruses.

Diagnosis:-Diagnosed by electron microscopy only, often very difficult because of small size.

Treatment:-don't use drugs because it is self-limiting so give only supportive care(Symptomatic treatment) mainly oral rehydration to correct electrolytes imbalance

Summary: Non-inflammatory: Viruses

	Norovirus (Norwalk) virus	Rotaviruses	Adenovirus	Astrovirus
Genome/ structure	Positive ss RNA, non-enveloped, Star of David	ds RNA (11 segments), non-enveloped, Wheel-like	ds DNA, non-enveloped, type40/41, fibers (penton)	positive ss RNA, non-enveloped
High risk	group settings, eg. cruise ships	Infants	Infants, older children, adults	Infants, elderly, immunocompromized
seasonality	Occurs year around	November-April peak	late fall and winter peak	Winter peak
Vaccine	no	Yes,	no	no

(5)Other Viruses

Other agents associated with gastrointestinal diseases include coronavirus-like agents, toroviruses, and some group A coxsackieviruses (the latter primarily cause gastrointestinal symptoms in severely immunocompromised patients).

==========

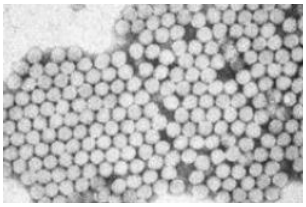
Viral hepatitis

Some note:-

1-Acute viral hepatitis: symptoms last less than 6 months but chronic state it will start after 6 month if virus is still in the liver

2-Hepatitis may be caused by at least five different viruses (A, B, C, D, E) Other viruses, such as Epstein–Barr virus and cytomegalovirus but different type of these viruses it may cause the same clinical picture(so use laboratory test to differentiate between it)

Hepatitis A (hepA)



Family:-is picornavirus

Characteristics of it:-+ssRNA viruses,naked virus,acid-stabile

Mode of replication:- it will replicates in the cytoplasm of liver,then excreted in bile and is then excreted in the faeces of infected persons for about 2 weeks before the onset of clinical illness and for up to 7 days after

Mode of transmission:-mainly Fecal-oral route&but also it might be saxually

Major host:-humen

People at risk:-children and young adults

epidemiology of the disease:-
1-Hepatitis A is the most common type of viral hepatitis and less dangerous than hepatitisB&C
2-There is no carrier state(unlike B&C)
3-Patients are most contagious in the 1 to 2 weeks prior to the onset of clinical disease
4-Travelers from developed countries who enter endemic areas are particularly susceptible

Incubation period :-10-50 days

Disease&symptoms:-
1-start with right upper abdominal quadrant(at site of liver)with fever and anorexia
2-after few days jundice,Dark urine and clay-colored stools
3-then hepatomegaly(enlarged&tendrensess in liver)
4-Chronic hepatitis is very rare(unlike hepatitis B)

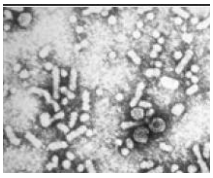
Diagnosis:-
1-Immune electron microscopic identification of the virus in fecal specimen
2-liver test(↑serum AST or ALT&↑serum bilirubin)
3-CBC----> leucopenia with a relative lymphocytosis&↑ESR
4-Viral markers: serum HAV IgG antibodies(and IgM in acute cases)

Treatment:-Almost all cases (99%) of hepatitis A are self-limiting. And recovery occurs in days to weeks without treatment or just give symptomatic treatment

Prevention:-
1-avoid contaminated food or water
2-Passive immunization with Immune serum globulin (ISG) during the incubation period
3-Active immunization with formalin-killed vaccines--->100% protective

---

Hepatitis B (hepB)



Family:-is Hepadna Virus Family

Characteristics of it:-it is smallest partially double-stranded DNA virus with a short, single stranded piece,enveloped,and it is contain 3 main antigen:1)hepatitis B core antigen (HBcAg)/2)pre-core-hepatitis B e antigen (HBeAg)/3)hepatitis B surface antigen on envelop (HBsAg)

Mode of replication:- Replicates in and outside the nuclus(without intergrate with host chromosome)

People at risk:-neonates,health care workers

Mode of transmission:-
1-intravenous route(e.g. by transfusion of infected blood)
2-Mainly sexually(because virus can found in seminal fluid)
3-by kissing(because virus can be found in saliva also)
4-Vertical transmission from mother to child

epidemiology of the disease:-
1-wide-spread world wide(400 million have chronic infection)
2-Needle stick injuries, has resulted in a higher risk of hepatitis B in medical personnel

Incubation period:-7-160 days(approximately 10 weeks)
Disease&symptoms:-

1-it is usually an asymptomatic or limited illness with right upper abdominal quadrant(at site of liver)with loss of appetite,fatigue, nausea,frank arthritis(swelling in joint) & rash
2-after few days increase cholestasis with jundice,Dark urine and clay-colored stools
3-then Fulminant hepatitis, leading to extensive liver necrosis(in 1% of cases)
4-chronic hepatitis(in10% of cases)
5-hepatocellular carcinoma in(25% of patients)

Diagnosis:-

1-liver test(AST, ALT, ALP, and total Bilirubin are elevated)
2-PCR(to detect DNA of virus)
3-liver biopsy(to detect cancer)
4-specific Serology(very very importent):-(as in picture)

- **HBsAg** - used as a general marker of infection.
- **HBsAb** - used to document recovery and/or immunity to HBV infection.
- **anti-HBc IgM** - marker of acute infection.
- **anti-HBcIgG** - past or chronic infection.
- **HBeAg** - indicates active replication of virus and therefore infectiveness.
- **Anti-Hbe** - virus no longer replicating. However, the patient can still be positive for HBsAg which is made by integrated HBV.
- **HBV-DNA** - indicates active replication of virus, more accurate than HBeAg especially in cases of escape mutants. Used mainly for monitoring response to therapy.

Treatment:-

1-For chronic hepatitis--->give interferon alpha &Adefovir (nucleotide analog of adenosine monophosphate)
2-Lamivudine(3TC)--->a potent inhibitor of HIV is also active versus hepatitis B virus both(but it has 25% resistance)

Prevention:-

1-avoid needles injuries
2-Vaccination(highly effective)
3-Hepatitis B Immunoglobulin-HBIG may be used to protect persons who are exposed to hepatitis B
4-screening of blood donors, blood and body fluid

Hepatitis D

Characteristics of it:(-)ssRNA,Enveloped,Circular genome&it is considered Co-infection because it cannot cause disease without HepB(Require HBsAg to be infectious)

هذا الفيروس اذا دخل جسم مريض ما عنده Hep B بكون وجوده وعدمه واحد بالجسم لانه هو بختبئ داخل الhep B عشان يقدر يدخل معه جوا الخلية ويعمل infection

People at risk:-Injection drug users&patient which do dialysis

Mode of transmission:-

1-mainly by blood transfusion or injection(like drugs)

2-Vertical transmission from mother to child

Disease&symptoms:-

1-Simultaneous delta and hepatitis B infection(Hepatitis B&D at the same time)----->fulminant hepatitis(acute liver necrosis) is much more common than with hepatitis B virus alone

2-Delta superinfection in those patients who have chronic hepatitis B --->developing chronic cirrhosis

Diagnosis:-

1-serological test for IgM antibodies against delta-virus in acute cases (IgM antibodies appear within 3 weeks of infection and persist for several weeks)

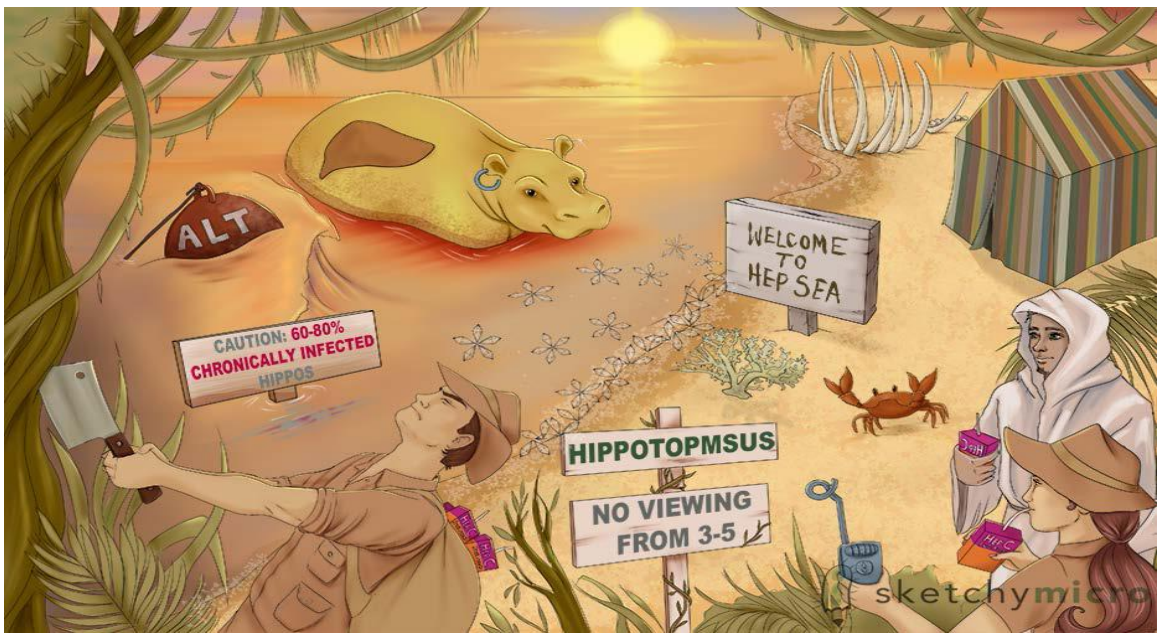
2-serological test for IgG antibodies against delta-virus in chronic cases(IgG antibodies persist for years)

3-PCR it can be useful

Treatment:-by interferon alpha

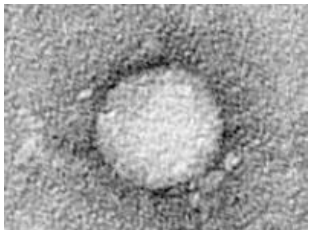
==========

Hepatitis C



Family:- flavivirus

Characteristics of it:non-segmented (+)ssRNA virus,enveloped,with antigenic variability of envelope proteins.



Mode of transmission:-
1-mainly by blood transfusion or needle sharing
2-May be also sexually transmitted

People at risk:-Injection drug users,health care worker,chronic hemodialysis patients and spouses(الازواج)

epidemiology of the disease:-
1-Hepatitis C was the major cause of post-transfusion hepatitis (because it is mainly transmitted by blood transfusion)
2-Needle sharing accounts for up to 40% of cases in USA
3-chronic carrier in 85% of cases

incubation period:-6-12 weeks

Disease&symptoms:-
1-**asymptomatic or mild ill with fever, malaise, headache, anorexia, vomiting, dark urine, jaundice**
2-60-80% of Hep C infections become chronic during 10-18years (unlike hep B which account for 10% only)
3-Primary cause of hepatocellular carcinoma and Cirrhosis

Diagnosis:-
1-in Acute phase:-↑serum IgM &ALT will rise and fall by 6 months
2-in chronic phase:-↑serum IgG
3-PCR

Treatment:-by combination therapy of interferon alpha +ribavirin+protease inhibitors

Prevention:-
1-Corticosteroids&passive immunization(ISG) or active vaccine are not beneficial
2-Avoidance of injection drug use and screening of blood products (reduced post-transfusion hepatitis by 80–90%-beneficial-)

You must look to the table in this link :-
https://drive.google.com/file/d/191_gjRLopXq-da_qFtRSEwR74kr-s0SA/view?usp=drivesdk